```
=> e sanders ira/au
E1            3     SANDERS IAN S/AU
E2            2     SANDERS ILSE M/AU
E3           46 --> SANDERS IRA/AU
E4            2     SANDERS IRL R III/AU
E5            2     SANDERS IRWIN T/AU
E6          498     SANDERS J/AU
E7           82     SANDERS J A/AU
E8            2     SANDERS J A C/AU
E9            1     SANDERS J A H/AU
E10           2     SANDERS J A M/AU
E11           2     SANDERS J ALAN/AU
E12          55     SANDERS J B/AU

=> s e3 and IgE
L1            1 "SANDERS IRA"/AU AND IGE

=> d

L1   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2004:467981  CAPLUS
DN   141:17606
TI   Use of a clostridial neurotoxin for the treatment of mammalian
     physiological reaction of IgE antibodies present upon contact
     with the corresponding antigen
IN   Sanders, Ira
PA   USA
SO   PCT Int. Appl., 28 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
     ---------------      ----   --------    --------------------    --------
PI   WO 2004048519         A2    20040610    WO 2003-US37286         20031120
     WO 2004048519         A3    20040701
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
             TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     CA 2507115            A1    20040610    CA 2003-2507115         20031120
     AU 2003295769         A1    20040618    AU 2003-295769          20031120
     EP 1565210            A2    20050824    EP 2003-786972          20031120
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
     US 2006008462         A1    20060112    US 2005-535504          20050518
PRAI US 2002-427749P       P     20021121
     WO 2003-US37286       W     20031120


=> s e3 and clostrid?
L2            4 "SANDERS IRA"/AU AND CLOSTRID?

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3             4 DUP REM L2 (0 DUPLICATES REMOVED)

=> d bib ab 1-
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YOU HAVE REQUESTED DATA FROM 4 ANSWERS - CONTINUE? Y/(N):y

L3 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:740453 CAPLUS
DN 141:236713
TI Cell membrane translocation of regulated SNARE inhibitors, compositions therefor, and methods for treatment of disease
IN Sanders, Ira
PA USA
SO PCT Int. Appl., 68 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004076634	A2	20040910	WO 2004-US5436	20040224
	WO 2004076634	A3	20050519		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1599213	A2	20051130	EP 2004-714146	20040224
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 2006153876	A1	20060713	US 2005-545872	20050817
PRAI	US 2003-449107P	P	20030224		
	WO 2004-US5436	W	20040224		

AB Compns. and methods of modulating cellular function and treatment of disease in mammals are disclosed which comprise locally administering a regulated SNARE inhibitor and a translocating agent to the mammal. Regulated SNARE inhibitors include clostridial neurotoxins, tetanus neurotoxin and their free light chain portions and IgA protease. Translocating agents include acids, encapsulating vectors, and transduction domains.

L3 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:467981 CAPLUS
DN 141:17606
TI Use of a clostridial neurotoxin for the treatment of mammalian physiological reaction of IgE antibodies present upon contact with the corresponding antigen
IN Sanders, Ira
PA USA
SO PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004048519	A2	20040610	WO 2003-US37286	20031120
	WO 2004048519	A3	20040701		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,				

BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

	CA 2507115	A1	20040610	CA 2003-2507115	20031120
	AU 2003295769	A1	20040618	AU 2003-295769	20031120
	EP 1565210	A2	20050824	EP 2003-786972	20031120
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 2006008462	A1	20060112	US 2005-535504	20050518
PRAI	US 2002-427749P	P	20021121		
	WO 2003-US37286	W	20031120		

AB A method is disclosed for blocking or reducing physiol. reaction in a mammal to the interaction of IgE antibodies present in the mammal upon contact with the corresponding antigen, by the administration to the mammal of a therapeutically effective amount of a neurotoxin derived from Clostridia sp.

L3 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:162795 CAPLUS
DN 140:193112
TI Treatment of holocrine gland dysfunction with clostridial neurotoxins
IN Sanders, Ira; Aquila, Rosemary
PA USA
SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004016763	A2	20040226	WO 2003-US25708	20030818
	WO 2004016763	A3	20040610		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2496005	A1	20040226	CA 2003-2496005	20030818
	AU 2003263860	A1	20040303	AU 2003-263860	20030818
	EP 1545207	A2	20050629	EP 2003-788573	20030818
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 2005220820	A1	20051006	US 2005-524304	20050208
PRAI	US 2002-404378P	P	20020819		
	WO 2003-US25708	W	20030818		

AB Methods of using clostridial toxins and other biol. agents to control holocrine gland dysfunction in humans is provided. In preferred embodiments, the methods provide beneficial effects in humans.

L3 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1995:988229 CAPLUS
DN 124:21809
TI Treatment of automatic nerve dysfunctions with botulinum toxin
IN Sanders, Ira; Shaari, Christopher M.
PA Mount Sinai School of Medicine of the City University of New York, USA
SO PCT Int. Appl., 39 pp.
CODEN: PIXXD2
DT Patent

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LA   English
FAN.CNT 1
     PATENT NO.            KIND   DATE        APPLICATION NO.          DATE
     ---------------       ----   --------    --------------------     --------
PI   WO 9528171             A1    19951026    WO 1995-US4558           19950413
         W:  JP
         RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
     EP 705106              A1    19960410    EP 1995-916366           19950413
         R:  DE, FR, GB, IT
PRAI US 1994-228132         A     19940415
     WO 1995-US4558         W     19950413
AB   There is disclosed a method for the control of autonomic nerve function in
     a mammal comprising administering a therapeutically effective amount of
     botulinum toxin to the mammal.  Preferred embodiments include
     administering the toxin to control the function of an autonomic nerve
     which contributes to at least one symptom of rhinorrhea, otitis media,
     excessive salivation, asthma, COPD, excessive stomach acid secretion,
     spastic colitis or excessive sweating.  For example, rhinorrhea can be
     treated by administering botulinum toxin onto the nasal mucosa or by
     injecting into the sphenopalatine ganglion.


=> s IgE and (clostrid?)
L4          118 IGE AND (CLOSTRID?)

=> s l4 and toxin?
L5           44 L4 AND TOXIN?

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6           31 DUP REM L5 (13 DUPLICATES REMOVED)

=> d bib ab 1-
YOU HAVE REQUESTED DATA FROM 31 ANSWERS - CONTINUE? Y/(N):y

L6   ANSWER 1 OF 31  CAPLUS  COPYRIGHT 2007 ACS on STN
AN   2007:817006  CAPLUS
DN   147:197358
TI   Stable therapeutic formulations
IN   Ameri, Mahmoud; Cormier, Michel J. N.; Sellers, Scott; Maa, Yuh-Fun
PA   Alza Corp., USA
SO   PCT Int. Appl., 50pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.            KIND   DATE        APPLICATION NO.          DATE
     ---------------       ----   --------    --------------------     --------
PI   WO 2007084247          A2    20070726    WO 2006-US49488          20061228
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN,
             KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK,
             MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO,
             RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT,
             TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
         RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE,
             IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ,
             CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH,
             GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY,
             KG, KZ, MD, RU, TJ, TM
     US 2007184096          A1    20070809    US 2006-617639           20061228
PRAI US 2005-754948P        P     20051228
AB   Compns. of and methods for formulating and delivering biol. active agent
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formulations having enhanced phys. stability, and wherein deterioration from the presence of oxygen and/or water is minimized and/or controlled, to yield a stable formulation are claimed. The compns. of and methods for formulating and delivering biol. active agents of the present invention further facilitate their incorporation into a biocompatible coating which can be employed to coat a stratum corneum piercing microprojection, or a plurality of stratum corneum piercing microprojections of a delivery device, for delivery of the biocompatible coating through the skin of a subject, thus providing an effective means of delivering the biol. active agents. A delivery device having stratum corneum piercing microprojections coated with a formulation of hPTH (1-34)was prepared The primary packaging for all dosages of the systems was a heat sealed foil pouch purged with nitrogen gas. The moisture and oxygen levels were substantially reduced in the packages.

L6 ANSWER 2 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:678518 CAPLUS
DN 145:123037
TI Engineered immunoglobulin domains with modified structural loop region to obtain antigen epitope binding and/or targeting property for analytical, diagnostic and therapeutic use
IN Rueker, Florian; Wozniak-Knopp, Gordana
PA Austria
SO PCT Int. Appl., 98 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2006072620	A1	20060713	WO 2006-EP50059	20060105
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	AU 2006204459	A1	20060713	AU 2006-204459	20060105
	EP 1699826	A1	20060913	EP 2006-703578	20060105
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU				
	EP 1752471	A1	20070214	EP 2006-121439	20060105
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
	EP 1772465	A1	20070411	EP 2006-11173	20060105
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, YU				
PRAI	US 2005-641144P	P	20050105		
	EP 2006-703578	A3	20060105		
	WO 2006-EP50059	W	20060105		

AB Method for engineering an Ig comprising at least one modification in a structural loop region of said Ig and determining the binding of said Ig to an epitope of an antigen, wherein the unmodified Ig does not significantly bind to said epitope, comprising the steps of: providing a nucleic acid encoding an Ig comprising at least one structural loop region, modifying

at least one nucleotide residue of at least one of said structural loop regions, transferring said modified nucleic acid in an expression system, expressing said modified Ig, contacting the expressed modified Ig with an epitope, and determining whether said modified Ig binds to said epitope, as well as modified Igs.

RE.CNT 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 3 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:654061 CAPLUS
DN 145:102141
TI Anti-toxin antibodies stabilized for oral delivery
IN Hansen, Genevieve; Demarest, Stephen J.
PA Diversa Corporation, USA
SO PCT Int. Appl., 263 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006071877	A2	20060706	WO 2005-US47100	20051222
WO 2006071877	A3	20070405		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
AU 2005321974	A1	20060706	AU 2005-321974	20051222
PRAI US 2004-639827P	P	20041227		
WO 2005-US47100	W	20051222		

AB The authors disclose engineering of therapeutic antibodies to increase their stability and resistance to proteases, e.g., in the digestive tract. Protease cleavage motifs are identified and subsequently modified to reduce or eliminate cleavage at that site. In one example, neutralizing antibodies, stabilized to low pH and pepsin degradation, were engineered against Clostridium difficile toxin A. In addition, the authors also disclose combinations of monoclonal antibodies that work synergistically to neutralize bacterial toxins.

L6 ANSWER 4 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2006:414826 BIOSIS
DN PREV200600422488
TI 1st Annual Conference of the Swiss Network on Horse Research, Avenches, SWITZERLAND.
AU Anonymous
SO Schweizer Archiv fuer Tierheilkunde, (APR 2006) Vol. 148, No. 4, pp. 200-212.
Meeting Info.: 1st Annual Conference of the Swiss Network on Horse Research. Avenches, SWITZERLAND. 20060412,.
CODEN: SATHAA. ISSN: 0036-7281.
DT Conference; (Meeting)
Conference; (Meeting Summary)
LA English
ED Entered STN: 23 Aug 2006
Last Updated on STN: 23 Aug 2006

AB This meeting, which focuses on animal husbandry, contains abstracts of 41 papers, written in English, French and German. Topics include the etiology and treatment of inflammatory airway disease and recurrent airway obstruction in horses, laparascopic surgery procedures, toxicoinfection in grazing horses, the role of T cells in insect bite hypersensitivity development, antibiotic treatment in foals and equine genetics.

L6 ANSWER 5 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:238846 CAPLUS
DN 142:309938
TI Re-targeted toxin conjugates
IN Foster, Keith; Chaddock, John; Penn, Charles
PA Health Protection Agency, UK
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005023309	A2	20050317	WO 2004-GB3904	20040913
	WO 2005023309	A3	20050915		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2004269979	A1	20050317	AU 2004-269979	20040913
	CA 2538619	A1	20050317	CA 2004-2538619	20040913
	EP 1667725	A2	20060614	EP 2004-768450	20040913
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK				
	JP 2007505094	T	20070308	JP 2006-525899	20040913
	US 2007184048	A1	20070809	US 2006-571515	20060907
PRAI	GB 2003-21344	A	20030911		
	WO 2004-GB3904	W	20040913		

AB The present invention provides a method for designing a re-targeted toxin conjugate for use in treating a medical condition or disease. Also provided, is the use of said conjugates in the manufacture of a medicament for treating medical conditions or diseases. The conjugates include a Targeting Moiety, which directs the conjugate to a desired target cell, and are characterized by a Targeting Moiety that increases exocytic fusion in the target cell. The present invention also provides methods for identifying agonists suitable for use as Targeting Moieties, and methods for preparing conjugates comprising said Targeting Moieties.

L6 ANSWER 6 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:614580 CAPLUS
DN 143:139175
TI Frequency-assisted transdermal agent delivery method and system
IN Chan, Keith T.; Cormier, Michel J. N.; Lin, WeiQi
PA USA
SO U.S. Pat. Appl. Publ., 24 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI	US 2005153873	A1	20050714	US 2004-971441	20041021
	AU 2004314416	A1	20050804	AU 2004-314416	20041021
	WO 2005069758	A2	20050804	WO 2004-US34923	20041021
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	BR 2004017757	A	20070410	BR 2004-17757	20041021
	JP 2007519446	T	20070719	JP 2006-549239	20041021
PRAI	US 2004-535275P	P	20040109		
	WO 2004-US34923	W	20041021		

AB The invention discloses an apparatus and method for transdermally delivering a biol. active agent comprising a delivery system having a microprojection member (or system) that includes a plurality of microprojections (or array thereof) that are adapted to pierce through the stratum corneum into the underlying epidermis layer, or epidermis and dermis layers, a formulation containing the biol. active agent and an oscillation-inducing device. In one embodiment, the biol. active agent is contained in a biocompatible coating that is applied to the microprojection member. In a further embodiment, the delivery system includes a gel pack having an agent-containing hydrogel formulation that is disposed on the microprojection member after application to the skin of a patient. In an alternative embodiment, the biol. active agent is contained in both the coating and the hydrogel formulation.

L6 ANSWER 7 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2005:524977 CAPLUS
DN 143:20047
TI Lipid rafts and clostridial toxins
IN Li, Shengwen; Aoki, Kei Roger
PA USA
SO U.S. Pat. Appl. Publ., 17 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2005129677	A1	20050616	US 2003-732703	20031210
	AU 2004315599	A1	20050825	AU 2004-315599	20041210
	CA 2549432	A1	20050825	CA 2004-2549432	20041210
	WO 2005077416	A2	20050825	WO 2004-US41235	20041210
	WO 2005077416	A3	20060713		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	EP 1691799	A2	20060823	EP 2004-821355	20041210
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, BA, HR, IS, YU

	JP 2007516257	T	20070621	JP 2006-543976	20041210
PRAI	US 2003-732703	A	20031210		
	WO 2004-US41235	W	20041210		

AB The present invention is directed to methods of altering the degree of internalization of a Clostridial toxin; methods of preventing or treating botulinum toxin intoxication; methods of treating metabolic disorders, muscular disorders, nervous system disorders, and/or pain conditions; methods of inhibiting the formation of lipid rafts on cell membranes; methods of treating a disease associated with lipid rafts; and methods of identifying a compound that alters the internalization of a Clostridial toxin.

L6 ANSWER 8 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:54068 CAPLUS
DN 144:306436
TI Recombinant expression of Clostridium tetani tetanus toxin monoclonal antibody light chain variable region and its applications
IN Gong, Jianghong; Li, Zhuoya
PA Gong, Xiaodi, Peop. Rep. China
SO Faming Zhuanli Shenqing Gongkai Shuomingshu, 33 pp.
CODEN: CNXXEV
DT Patent
LA Chinese
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	CN 1634991	A	20050706	CN 2003-10122187	20031230
PRAI	CN 2003-10122187		20031230		

AB The present invention relates to recombinant expression of Clostridium tetani tetanus toxin monoclonal antibody light chain variable region. The monoclonal antibody comprises the variable region of antibody and/or constant region of light chain or heavy chain of antibody. The humanized monoclonal antibody is prepared by gene recombination technique and can be used for diagnosis, prevention, and treatment of Clostridium tetani infection.

L6 ANSWER 9 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2005:20849 BIOSIS
DN PREV200500024041
TI Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof.
AU Bigalke, Hans [Inventor, Reprint Author]; Frevert, Jurgen [Inventor]
CS Hannover, DE, USA
ASSIGNEE: BioteCon Therapeutics GmbH, Potsdam, Germany
PI US 6822076 20041123
SO Official Gazette of the United States Patent and Trademark Office Patents, (Nov 23 2004) Vol. 1288, No. 4. http://www.uspto.gov/web/menu/patdata.html . e-file.
ISSN: 0098-1133 (ISSN print).
DT Patent
LA English
ED Entered STN: 29 Dec 2004
Last Updated on STN: 29 Dec 2004
AB A hybrid protein contains a protein that binds to a receptor of mastocytes and basophils and is endocyted by them. The protein can be IgE; IgE fragment; IgE Fc fragment; antibody against IgE receptor of mastocytes and basophils; fragment of the antibody against the IgE receptor of mastocytes and basophils; antibody against mastocyte specific potassium channel; and mast cell degranulating peptide. The hybrid protein also contains a protease cleaving proteins of the secretion process of the mastocytes and basophils so as to inhibit the

secretion process without killing the mastocytes and basophils. The protease can be light chain Clostridium botulinum toxin ; proteolytically active fragment of the light chain of a Clostridium botulinum toxin containing an amino acid sequence His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His wherein Xaa is an amino acid; light chain of the tetanus toxin; proteolytically active fragment of the light chain of the tetanus toxin containing His-Asp-Leu-lIe-His-Val-Leu-His; IgA protease of Neisseria gonorrhoeae; and proteolytic domain of the IgA protease of Neisseria gonorrhoeae.

L6 ANSWER 10 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:467981 CAPLUS
DN 141:17606
TI Use of a clostridial neurotoxin for the treatment of mammalian physiological reaction of IgE antibodies present upon contact with the corresponding antigen
IN Sanders, Ira
PA USA
SO PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2004048519	A2	20040610	WO 2003-US37286	20031120
	WO 2004048519	A3	20040701		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	CA 2507115	A1	20040610	CA 2003-2507115	20031120
	AU 2003295769	A1	20040618	AU 2003-295769	20031120
	EP 1565210	A2	20050824	EP 2003-786972	20031120
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	US 2006008462	A1	20060112	US 2005-535504	20050518
PRAI	US 2002-427749P	P	20021121		
	WO 2003-US37286	W	20031120		

AB A method is disclosed for blocking or reducing physiol. reaction in a mammal to the interaction of IgE antibodies present in the mammal upon contact with the corresponding antigen, by the administration to the mammal of a therapeutically effective amount of a neurotoxin derived from Clostridia sp.

L6 ANSWER 11 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2004:609728 CAPLUS
DN 141:156089
TI Amended recombinant microbial cells encoding γ interferon as antiviral agent, adjuvant and vaccine accelerant
IN Gaertner, Frank H.; Lee, Stacey Lynn; Shutter, Robert W.
PA Dow Agrosciences LLC, USA
SO U.S. Pat. Appl. Publ., 34 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI	US 2004146484	A1	20040729	US 2003-681540	20031007
	CA 2501690	A1	20041014	CA 2003-2501690	20031007
	WO 2004087864	A2	20041014	WO 2003-US31815	20031007
	WO 2004087864	A3	20041223		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003304025	A1	20041025	AU 2003-304025	20031007
	EP 1549346	A2	20050706	EP 2003-816565	20031007
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	BR 2003014542	A	20050726	BR 2003-14542	20031007
	CN 1723040	A	20060118	CN 2003-80105335	20031007
	JP 2006510388	T	20060330	JP 2004-570237	20031007
	NZ 539207	A	20061027	NZ 2003-539207	20031007
	US 2006040352	A1	20060223	US 2005-38901	20050118
	ZA 2005002746	A	20051013	ZA 2005-2746	20050405
	MX 2005PA03692	A	20050930	MX 2005-PA3692	20050407
	US 2006234346	A1	20061019	US 2006-400840	20060407
PRAI	US 2002-417124P	P	20021008		
	US 2003-681540	A2	20031007		
	WO 2003-US31815	W	20031007		
	US 2004-537148P	P	20040116		
	US 2004-564798P	P	20040422		
	US 2005-38901	A1	20050118		

AB The present invention provides active cytokine and/or chemokine compns., as well as inexpensive means for the production, amended-cell encasement of active cytokine and/or chemokine compns., processing, and delivery of active cytokine and/or chemokine compns. The subject invention also provides methods of treatment and methods of accelerating an immune response comprising the administration of amended recombinant cell (ARC) containing cytokine and/or chemokine compns. to animals or humans. In example, the amended recombinant cell was Pseudomonas fluorescens, and the cytokine was bovine γ interferon.

L6 ANSWER 12 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:300440 CAPLUS
DN 138:319681
TI Genetically-detoxified pertussis holotoxin as proteinaceous adjuvant
IN Gajewczyk, Diane M.; Boux, Heather A.; Novak, Anton; Klein, Michel H.
PA Can.
SO U.S. Pat. Appl. Publ., 25 pp., Cont.-in-part of U.S. Ser. No. 258,228.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2003072774	A1	20030417	US 1995-481878	19950607
	CA 2192454	A1	19951221	CA 1995-2192454	19950608
	EP 1149588	A1	20011031	EP 2001-201598	19950608
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
	EP 1149589	A1	20011031	EP 2001-201610	19950608
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE				
	PT 764029	T	20021031	PT 1995-924122	19950608
	ES 2179105	T3	20030116	ES 1995-924122	19950608

PRAI US 1994-258228 A2 19940610
EP 1995-924122 A3 19950608

AB A modulated immune response to an antigen is achieved by coadministering the antigen and a genetically-detoxified pertussis holotoxin, particularly one retaining its immunogenicity, to a host. The modulated immune response enables immunogenic compns., including multivalent pediatric vaccines, such as DTP, to be provided which produce a modulated immune response in the absence of extrinsic adjuvants, such as alum. The adjuvanting effect achieved by the genetically-detoxified pertussis holotoxin enables at least the same level of a modulated immune response to a non-Bordetella antigen to be achieved as previously attained by alum, without the undesirable side effects thereof. Modifications are possible within the scope of the disclosed invention.

L6 ANSWER 13 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:241912 CAPLUS
DN 138:265639
TI Inhibiting the degranulation in mastocytes using a hybrid protein comprising a receptor-binding protein fused to a protease cleaving a protein of the secretion process
IN Bigalke, Hans; Frevert, Jurgen
PA Biotecon Gesellschaft Fur Biotechnologische Entwicklung Und Consulting Mbh, Germany
SO U.S. Pat. Appl. Publ., 7 pp., Cont.-in-part of U.S. Ser. No. 700,540.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 2

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI US 2003059912	A1	20030327	US 2002-64903	20020827
US 6822076	B2	20041123		
WO 9958571	A2	19991118	WO 1999-EP3272	19990512
WO 9958571	A3	20000203		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI DE 1998-19821285 A 19980513
WO 1999-EP3272 W 19990512
US 2001-700540 A2 20010119

AB A hybrid protein is provided containing a protein that binds to a receptor of mastocytes and basophils and is endocytosed by them. The protein can be IgE, IgE fragment, IgE Fc fragment, antibody against the IgE receptor of mastocytes and basophils, a fragment of the antibody against the IgE receptor of mastocytes and basophils, an antibody against mastocyte-specific potassium channel, or mast cell degranulating peptide. The hybrid protein also contains a protease which cleaves proteins of the secretion process of the mastocytes and basophils so as to inhibit the secretion process without killing the mastocytes and basophils. The protease can be the light chain of Clostridium botulinum toxin or its proteolytic fragments containing a His-Xaa-Xaa-Xaa-His-Xaa-Xaa-His sequence, the light chain of the tetanus toxin or proteolytically active fragment of the light chain containing His-Asp-Leu-Ile-His-Val-Leu-His, or an IgA protease of Neisseria gonorrhoeae and its proteolytic domain. Thus, a hybrid protein comprising IgE fused to the light chain of either Clostridium botulinum toxin or tetanus toxin prevents allergic shock caused by dying mastocytes.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 14 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 1
AN 2003:190213 BIOSIS
DN PREV200300190213
TI Effects of large clostridial cytotoxins on activation of RBL 2H3-hm1 mast cells indicate common and different roles of Rac in FcepsilonRI and M1-receptor signaling.
AU Djouder, Nabil; Aneiros, Eduardo; Cavalie, Adolfo; Aktories, Klaus [Reprint Author]
CS Institut fuer Experimentelle und Klinische Pharmakologie und Toxikologie, der Albert-Ludwigs-Universitaet Freiburg, Albertstrasse 25, Otto Krayer Haus, D-79104, Freiburg, Germany
klaus.aktories@pharmakol.uni-freiburg.de
SO Journal of Pharmacology and Experimental Therapeutics, (March 2003) Vol. 304, No. 3, pp. 1243-1250. print.
ISSN: 0022-3565 (ISSN print).
DT Article
LA English
ED Entered STN: 16 Apr 2003
Last Updated on STN: 16 Apr 2003
AB Using Rho GTPases-inhibiting clostridial cytotoxins, we showed recently in RBL cells that the GTPase Rac is involved in FcepsilonRI (high-affinity receptor for IgE) signaling and receptor-mediated calcium mobilization, including influx via calcium release-activated calcium channels. Here, we studied the role of Rho GTPases in muscarinic M1 receptor signaling in RBL 2H3-hm1 cells. Clostridium difficile toxin B, which inactivates Rho, Rac, and Cdc42, and Clostridium sordellii lethal toxin, which inhibits Rac but not Rho, blocked M1-mediated exocytosis, indicating that Rac but not Rho is involved in the regulation of receptor-mediated exocytosis. Although antigen-induced FcepsilonRI stimulation caused tyrosine phosphorylation of the Rac guanine nucleotide exchange factor Vav, M1 stimulation by carbachol activated Rac independently of Vav. The Rac-inactivating toxins blocked M1 receptor-induced membrane translocation of the pleckstrin homology domain of protein kinase B, which is a phosphoinositide 3-kinase effector. The M1-induced calcium release from internal stores was not affected by toxin B; however, the subsequent calcium influx from the extracellular space was inhibited. The data suggest that besides capacitative calcium entry, the M1 signaling pathway activates further calcium entry channels with mechanisms that are not affected by the inhibition of Rac.

L6 ANSWER 15 OF 31 LIFESCI COPYRIGHT 2007 CSA on STN
AN 2003:5772 LIFESCI
TI Enhanced sensitisation of mice with diphtheria tetanus acellular pertussis vaccine to local swelling reaction to the booster immunisation
AU Yamamoto, Akihiko; Nagata, Noriyo; Ochiai, Masaki; Kataoka, Michiyo; Toyoizumi, Hiromi; Okada, Kenji; Horiuchi, Yoshinobu
CS Department of Bacterial Pathogenesis and Infection Control, National Institute of Infectious Diseases, 4-7-1 Gakuen, Musashimurayama, Tokyo 208-0011, Japan; E-mail: yama-aki@nih.go.jp
SO Vaccine, (20020819) vol. 20, no. 25-26, pp. 3088-3094.
ISSN: 0264-410X.
DT Journal
FS F; J
LA English
SL English
AB Severe local swelling has been regarded as a serious safety problem for the booster immunisations of diphtheria tetanus acellular pertussis combined (DTaP) vaccine and DT combined toxoids (DT-td). We attempted to search for the factor of DTaP vaccines possibly contributing to the enhanced local reaction by using the mouse hind paw swelling reaction.

Mice were immunised intramuscularly with DTaP vaccine twice at 1-month interval and were challenged their hind paw with one of the antigens of DTaP vaccine 2 weeks later. D-td was shown to elicit the strongest swelling among the vaccine antigens. No causal relationship was found between the swelling and the level of immunoglobulin G (IgG) or IgE in mice. Residual pertussis toxin (PT) activity of DTaP vaccines for immunisation was shown to play a role in the enhanced sensitisation of mice to the D-td-related hind paw swelling.

L6 ANSWER 16 OF 31 MEDLINE on STN
AN 2001298444 MEDLINE
DN PubMed ID: 11380253
TI Phospholipases stimulate secretion in RBL mast cells.
AU Cohen J S; Brown H A
CS Department of Molecular Medicine, Veterinary Medical Center, and Field of Biochemistry, Molecular and Cellular Biology, Cornell University, Ithaca, New York 14853-6401, USA.
NC GM58516 (NIGMS)
SO Biochemistry, (2001 Jun 5) Vol. 40, No. 22, pp. 6589-97.
Journal code: 0370623. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, NON-P.H.S.)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 200108
ED Entered STN: 20 Aug 2001
Last Updated on STN: 20 Aug 2001
Entered Medline: 16 Aug 2001
AB Roles for glycerophospholipids in exocytosis have been proposed, but remain controversial. Phospholipases are stimulated following the activation of the high-affinity receptor for immunoglobulin E (IgE) in mast cells. To study the biochemical sequelae that lead to degranulation, broken cell systems were employed. We demonstrate that the addition of three distinct types of exogenous phospholipases (i.e., bcPLC, scPLD, and tfPLA(2)), all of which hydrolyze phosphatidylcholine (PC), trigger degranulation in permeabilized RBL-2H3 cells, a mucosal mast cell line. Production of bioactive lipids by these phospholipases promotes release of granule contents through the plasma membrane and acts downstream of PKC, PIP(2), and Rho subfamily GTPases in regulated secretion. These exogenous phospholipase-induced degranulation pathways circumvent specific factors activated following stimulation of the IgE receptor as well as in ATP- and GTP-dependent intracellular pathways. Taken together, these results suggest that regulated secretion may be achieved in vitro in the absence of cytosolic factors via phospholipase activation and that products of PC hydrolysis can promote exocytosis in mast cells.

L6 ANSWER 17 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 2
AN 2001:100080 CAPLUS
DN 134:264878
TI Rac and phosphatidylinositol 3-kinase regulate the protein kinase B in FcεRI signaling in RBL 2H3 mast cells
AU Djouder, Nabil; Schmidt, Gudula; Frings, Monika; Cavalie, Adolfo; Thelen, Marcus; Aktories, Klaus
CS Institut fur Pharmakologie und Toxikologie der Universitat Freiburg, Freiburg, D-79104, Germany
SO Journal of Immunology (2001), 166(3), 1627-1634
CODEN: JOIMA3; ISSN: 0022-1767
PB American Association of Immunologists
DT Journal
LA English

AB FcεRI signaling in rat basophilic leukemia cells depends on phosphatidylinositol 3-kinase (PI3-kinase) and the small GTPase Rac. Here, the authors studied the functional relation among PI3-kinase, its effector protein kinase B (PKB), and Rac using inhibitors of PI3-kinase and toxins inhibiting Rac. Wortmannin, an inhibitor of PI3-kinase, blocked FcεRI-mediated tyrosine phosphorylation of phospholipase Cγ, inositol phosphate formation, calcium mobilization, and secretion of hexosaminidase. Similarly, Clostridium difficile toxin B, which inactivates all Rho GTPases including Rho, Rac and Cdc42, and Clostridium sordellii lethal toxin, which inhibits Rac (possibly Cdc42) but not Rho, blocked these responses. Stimulation of the FcεRI receptor induced a rapid increase in the GTP-bound form of Rac. Whereas toxin B inhibited the Rac activation, PI3-kinase inhibitors (wortmannin and LY294002) had no effect on activation of Rac. In line with this, wortmannin had no effect on tyrosine phosphorylation of the guanine nucleotide exchange factor Vav. Wortmannin, toxin B, and lethal toxin inhibited phosphorylation of PKB on Ser473. Similarly, translocation of the pleckstrin homol. domain of PKB tagged with the green fluorescent protein to the membrane, which was induced by activation of the FcεRI receptor, was blocked by inhibitors of PI3-kinase and Rac inactivation. Our results indicate that in rat basophilic leukemia cells Rac and PI3-kinase regulate PKB and suggest that Rac is functionally located upstream and/or parallel of PI3-kinase/PKB in FcεRI signaling.
RE.CNT 65 THERE ARE 65 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 18 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2000:445738 CAPLUS
DN 133:188989
TI Inhibition of calcium release-activated calcium current by Rac/Cdc42-inactivating clostridial cytotoxins in RBL cells
AU Djouder, Nabil; Prepens, Ulrike; Aktories, Klaus; Cavalie, Adolfo
CS Institut fur Pharmakologie und Toxikologie der Universitat Freiburg, Freiburg, D-79104, Germany
SO Journal of Biological Chemistry (2000), 275(25), 18732-18738
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB Using large clostridial cytotoxins as tools, the role of Rho GTPases in the activation of RBL 2H3 hm1 cells was studied. Clostridium difficile toxin B, which glucosylates Rho, Rac, and Cdc42, and Clostridium sordellii lethal toxin , which glucosylates Rac and Cdc42 but not Rho, inhibited the release of hexosaminidase from RBL cells mediated by the high affinity antigen receptor (FcεRI). Addnl., toxin B and lethal toxin inhibited the intracellular Ca2+ mobilization induced by FcεRI stimulation and thapsigargin, mainly by reducing the influx of extracellular Ca2+. In patch clamp recordings, toxin B and lethal toxin inhibited the calcium release-activated calcium current by .apprx.45%. Calcium release-activated calcium current, the receptor-stimulated Ca2+ influx, and secretion were inhibited neither by the Rho-ADP-ribosylating C3-fusion toxin C2IN-C3 nor by the actin-ADP-ribosylating Clostridium botulinum C2 toxin. The data indicate that Rac and Cdc42 but not Rho are not only involved in late exocytosis events but are also involved in Ca2+ mobilization most likely by regulating the Ca2+ influx through calcium release-activated calcium channels activated via FcεRI receptor in RBL cells.
RE.CNT 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 19 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN

AN 1999:736768 CAPLUS
DN 131:332099
TI Hybrid protein for inhibiting the degranulation of mastocytes and the use thereof
IN Bigalke, Hans; Frevert, Jurgen
PA Biotecon Gesellschaft fur Biotechnologische Entwicklung und Consulting m.b.H, Germany
SO PCT Int. Appl., 22 pp.
CODEN: PIXXD2
DT Patent
LA German
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9958571	A2	19991118	WO 1999-EP3272	19990512
	WO 9958571	A3	20000203		
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2331274	A1	19991118	CA 1999-2331274	19990512
	AU 9942605	A	19991129	AU 1999-42605	19990512
	AU 755513	B2	20021212		
	BR 9910359	A	20010109	BR 1999-10359	19990512
	EP 1084146	A2	20010321	EP 1999-950347	19990512
	EP 1084146	B1	20021113		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
	HU 200103601	A2	20020128	HU 2001-3601	19990512
	JP 2002514659	T	20020521	JP 2000-548373	19990512
	AT 227739	T	20021115	AT 1999-950347	19990512
	PT 1084146	T	20030228	PT 1999-950347	19990512
	ES 2187200	T3	20030516	ES 1999-950347	19990512
	RU 2214420	C2	20031020	RU 2000-131217	19990512
	CZ 294376	B6	20041215	CZ 2000-4161	19990512
	NO 2000005637	A	20001108	NO 2000-5637	20001108
	MX 2000PA11148	A	20030422	MX 2000-PA11148	20001113
	HK 1036994	A1	20030221	HK 2001-106685	20010921
	US 2003059912	A1	20030327	US 2002-64903	20020827
	US 6822076	B2	20041123		
PRAI	DE 1998-19821285	A	19980513		
	WO 1999-EP3272	W	19990512		
	US 2001-700540	A2	20010119		

AB The invention relates to a hybrid protein comprising or comprised of (i) a known protein which binds to mastocytes and/or basophils in a known manner and/or is absorbed thereby, and of (ii) a protease which splits one or more proteins of the secretory apparatus of the mastocytes and/or basophils. The examples discuss the synthesis of these hybrid proteins using expression vectors expressed in E. coli.

L6 ANSWER 20 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 3
AN 1999:692 BIOSIS
DN PREV199900000692
TI Fc receptor-mediated phagocytosis requires CDC42 and Rac1.
AU Massol, Philippe; Montcourrier, Philippe; Guillemot, Jean-Claude; Chavrier, Philippe [Reprint author]
CS Cent. Immunol. INSERM-CNRS Marseille-Luminy, Case 906, 13288 Marseille Cedex 9, France
SO EMBO (European Molecular Biology Organization) Journal, (Nov. 2, 1998) Vol. 17, No. 21, pp. 6219-6229. print.

CODEN: EMJODG. ISSN: 0261-4189.
DT Article
LA English
ED Entered STN: 11 Jan 1999
Last Updated on STN: 11 Jan 1999
AB At the surface of phagocytes, antibody-opsonized particles are recognized by surface receptors for the Fc portion of immunoglobulins (FcRs) that mediate their capture by an actin-driven process called phagocytosis which is poorly defined. We have analyzed the function of the Rho proteins Rac1 and CDC42 in the high affinity receptor for IgE (FcepsilonRI)-mediated phagocytosis using transfected rat basophil leukemia (RBL-2H3) mast cells expressing dominant inhibitory forms of CDC42 and Rac1. Binding of opsonized particles to untransfected RBL-2H3 cells led to the accumulation of F-actin at the site of contact with the particles and further, to particle internalization. This process was inhibited by Clostridium difficile toxin B, a general inhibitor of Rho GTP-binding proteins. Dominant inhibition of Rac1 or CDC42 function severely inhibited particle internalization but not F-actin accumulation. Inhibition of CDC42 function resulted in the appearance of pedestal-like structures with particles at their tips, while particles bound at the surface of the Rac1 mutant cell line were enclosed within thin membrane protrusions that did not fuse. These phenotypic differences indicate that Rac1 and CDC42 have distinct functions and may act cooperatively in the assembly of the phagocytic cup. Inhibition of phagocytosis in the mutant cell lines was accompanied by the persistence of tyrosine-phosphorylated proteins around bound particles. Phagocytic cup closure and particle internalization were also blocked when phosphotyrosine dephosphorylation was inhibited by treatment of RBL-2H3 cells with phenylarsine oxide, an inhibitor of protein phosphotyrosine phosphatases. Altogether, our data show that Rac1 and CDC42 are required to coordinate actin filament organization and membrane extension to form phagocytic cups and to allow particle internalization during FcR-mediated phagocytosis. Our data also suggest that Rac1 and CDC42 are involved in phosphotyrosine dephosphorylation required for particle internalization.

L6 ANSWER 21 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1998:363180 CAPLUS
DN 129:91616
TI Effects of toxin A from Clostridium difficile on mast cell activation and survival
AU Calderon, Gloria M.; Torres-Lopez, Javier; Lin, Tong-Jun; Chavez, Bibiana; Hernandez, Manuel; Munoz, Onofre; Befus, A. Dean; Enciso, J. Antonio
CS UIMEIP, Hospital de Pediatra, CMN Siglo XXI, IMSS, Mexico City, 06725, Mex.
SO Infection and Immunity (1998), 66(6), 2755-2761
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB Toxins A and B from C. difficile are the main cause of antibiotic-associated diarrhea and pseudomembranous colitis. They cause fluid accumulation, necrosis, and a strong inflammatory response when inoculated in intestinal loops. Since mast cells are a rich source of inflammatory mediators, abundant in the gut, and known to be involved in C. difficile-induced enteritis, the authors studied the in vitro effect of toxin A on isolated mast cells. Normal rats sensitized by infection with Nippostrongilus brasiliensis were used to isolate peritoneal mast cells (PMC). PMC from naive rats were stimulated with calcium ionophore A23187 as a model of antigen-independent activation, and PMC from sensitized rats were stimulated with N. brasiliensis antigens to study IgE-dependent mast cell activation. After 4 h, toxin A did not induce the release of nitric oxide or histamine in naive PMC. However, 10 ng of toxin per mL caused a significant release of tumor necrosis factor α (TNF-α). In contrast, 1

μg of toxin per mL inhibited antigen or A23187-induced histamine release by PMC. Toxin A at 1 μg/mL for 4 h caused the disruption of actin which aggregated in the cytoplasm and around the nucleus. After 24 h, chromatin condensation, cytoplasmic blebbing, and apoptotic-like vesicles were observed; DNA fragmentation was documented also. These results suggest that mast cells may participate in the initial inflammatory response to C. difficile infection by releasing TNF-α upon interaction with toxin A. However, longer exposure to toxin A affects the release of inflammatory mediators, perhaps because of the alteration of the cytoskeleton and induction of apoptosis. The impaired functions and survival of mast cells by C. difficile toxin A could hamper the capacity of these cells to counteract the infection, thus prolonging the pathogenic effects of C. difficile toxins.

RE.CNT 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 22 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN DUPLICATE 4
AN 1998:137930 CAPLUS
DN 128:267074
TI Influence of Clostridium botulinum C2 toxin on FcεRI-mediated secretion and tyrosine phosphorylation in RBL cells
AU Prepens, Ulrike; Barth, Holger; Wilting, Jorg; Aktories, K.
CS Institut fur Pharmakologie und Toxikologie der Albert-Ludwigs-Universitat Freiburg, Hermann-Herder-Strasse 5, Freiburg, D-79104, Germany
SO Naunyn-Schmiedeberg's Archives of Pharmacology (1998), 357(3), 323-330
CODEN: NSAPCC; ISSN: 0028-1298
PB Springer-Verlag
DT Journal
LA English
AB The authors studied the effects of the binary Clostridium botulinum C2 toxin on stimulated [3H]serotonin release and protein tyrosine phosphorylation in RBL 2H3 hml cells. Actin was specifically ADP-ribosylated by C2 toxin in intact cells resulting in a 2-3 fold increase in antigen- or calcium ionophore (A23187)-induced degranulation. The effects of C2 toxin were time- and concentration-dependent. Toxin treatment, which dramatically changes the morphol. of RBL cells, was not sufficient to induce mediator release in the absence of activators of secretion. Antigen- and A23187-stimulated tyrosine phosphorylation of 60-80 kDa and 110-120 kDa proteins was reduced or blocked after C2 toxin incubation. Treatment of RBL cells with the tyrosine phosphatase inhibitor pervanadate reversed the inhibitory effect of C2 toxin on stimulated protein tyrosine phosphorylation indicating activation of phosphatases by C2 toxin. The data indicate that disassembly of the actin cytoskeleton by C2 toxin facilitates FcεRI-mediated signal-secretion coupling and suggest a role of the actin cytoskeleton in phosphatase regulation in RBL cells.

RE.CNT 59 THERE ARE 59 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 23 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 5
AN 1998:272096 BIOSIS
DN PREV199800272096
TI Role of RHO family GTP-binding proteins in IgE receptor-mediated phospholipase D activation in mast cells.
AU Ojio, Katsuhiro [Reprint author]; Banno, Yoshiko; Hayakawa, Kazuki; Ito, Yatsuji; Kato, Naoki; Watanabe, Kunitomo; Miyata, Hideo; Nozawa, Yoshinori
CS Dep. Otolaryngol., Gifu Univ. Sch. Med., Tsukasamachi 40, Gifu 500-9705, Japan
SO Biomedical Research (Tokyo), (Feb., 1998) Vol. 19, No. 1, pp. 53-63. print.
CODEN: BRESD5. ISSN: 0388-6107.

DT Article
LA English
ED Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998
AB To investigate the role of Rho family proteins in antigen-mediated phospholipase D (PLD) activation in cultured rat basophilic leukemia (RBL-2H3) mucosal mast cells, we used two toxins, Clostridium difficile toxin B (toxin B) and Clostridium botulinum C3 toxin (C3 toxin), which inhibit Rho family proteins by monoglucosylation and ADP-ribosylation, respectively. Pretreatment with toxin B caused rounding-out of RBL-2H3 cells, strong inhibition of antigen-induced PLD activation, and also a complete blockage of serotonin secretion. By contrast, C3 toxin was without effect on both PLD activation and serotonin secretion, although RhoA was markedly ADP-ribosylated. Recombinant ADP-ribosylation factor (Arf) stimulated the PLD activity in isolated membranes in a dose-dependent manner, and 4beta-phorbol 12-myristate 13-acetate (PMA) pretreatment of cells potentiated this recombinant Arf effect. The recombinant Arf- and PMA-stimulated PLD activities were partially inhibited by pretreatment with toxin B but not by C3 toxin. Stimulation of RBL cells with antigen induced translocation to membranes of factors involving PLD activation, e.g. protein kinase C (PKC) (alpha, beta2, delta6, epsilon) isozymes, Cdc42 and Arf, but not RhoA. These results suggest that the membrane-translocation of Cdc42 plays a major role in antigen-induced PLD activation in RBL cells and also that the translocated Arf and PKCs exert a co-operative effect for PLD activation.

L6 ANSWER 24 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 6
AN 1997:488094 BIOSIS
DN PREV199799787297
TI Oral tolerance to ovalbumin in mice: Induction and long-term persistence unaffected by Staphylococcus aureus enterotoxin B and Clostridium perfringens type A enterotoxin.
AU Gaboriau-Routhiau, Valerie [Reprint author]; Moreau, Marie-Christine
CS UEPSD, Bat. 440, CRJ INRA, 78352 Jouy-en-Josas Cedex, France
SO Pediatric Research, (1997) Vol. 42, No. 4, pp. 503-508.
CODEN: PEREBL. ISSN: 0031-3998.
DT Article
LA English
ED Entered STN: 7 Nov 1997
Last Updated on STN: 7 Nov 1997
AB Oral administration of dietary antigen (Ag) results in the systemic Ag-specific immunologic unresponsiveness termed oral tolerance. Its induction is of importance in the young where numerous symptoms are associated with IgE-mediated food hypersensitivity reactions. Two related enterotoxins, cholera toxin and Escherichia coli heat-labile enterotoxin, have been shown to abrogate oral tolerance (i.e. IgG and IgE antibody (Ab) unresponsiveness) to an unrelated and simultaneously fed Au However, a critical role has been suggested for the gut flora in recovery of a hyporesponsive state. The purpose of the present study was to investigate whether the Staphylococcus aureus enterotoxin B (SEB) and Clostridium perfringens type A enterotoxin (CPE), involved in many diarrheas, could affect the induction and long-term persistence of oral tolerance to ovalbumin (OVA). Using conventional and germ-free mice fed once or twice with enterotoxin plus OVA, we investigated the possible role of the indigenous gut flora. In addition, we tested the influence of CPE synthesized in vivo in the digestive tract of gnotobiotic mice on the induction of OVA-specific oral tolerance. Mice were immunized intraperitoneally with OVA twice, and IgG and IgE Ab levels were measured by ELISA. Neither SEB nor CPE, orally given or synthesized in vivo (CPE), prevented the induction of oral tolerance to OVA. Moreover, the IgG Ab unresponsiveness persisted over 2

mo in the conventional mice fed with toxin plus OVA as also observed in the OVA controls. The results indicate that, independent of the out flora's influence, SEB and CPE did not affect the induction and long-term persistence of oral tolerance to co-ingested Ag.

L6 ANSWER 25 OF 31 MEDLINE on STN
AN 96205904 MEDLINE
DN PubMed ID: 8631752
TI Inhibition of Fc epsilon-RI-mediated activation of rat basophilic leukemia cells by Clostridium difficile toxin B (monoglucosyltransferase).
AU Prepens U; Just I; von Eichel-Streiber C; Aktories K
CS Institut fur Pharmakologie und Toxikologie der Albert-Ludwigs-Universitat Freiburg, Germany.
SO The Journal of biological chemistry, (1996 Mar 29) Vol. 271, No. 13, pp. 7324-9.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199607
ED Entered STN: 15 Jul 1996
Last Updated on STN: 6 Feb 1998
Entered Medline: 3 Jul 1996
AB Treatment of rat basophilic leukemia (RBL) 2H3-hm1 cells with Clostridium difficile toxin B (2 ng/ml), which reportedly depolymerizes the actin cytoskeleton, blocked [3H]serotonin release induced by 2,4-dinitrophenyl-bovine serum albumin, carbachol, mastoparan, and reduced ionophore A23187-stimulated degranulation by about 55-60%. In lysates of RBL cells, toxin B 14C-glucosylated two major and one minor protein. By using two-dimensional gel electrophoresis and immunoblotting, RhoA and Cdc42 were identified as protein substrates of toxin B. In contrast to toxin B, Clostridium botulinum transferase C3 that selectively inactivates RhoA by ADP-ribosylation did not inhibit degranulation up to a concentration of 150 microg/ml. Antigen-stimulated tyrosine phosphorylation of a 110-kDa protein was inhibited by toxin B as well as by the phosphatidylinositol 3-kinase inhibitor wortmannin. Depolymerization of the microfilament cytoskeleton of RBL cells by C. botulinum C2 toxin or cytochalasin D resulted in an increased [3H]serotonin release induced by antigen, carbachol, mastoparan, or by calcium ionophore A23187, but without affecting toxin B-induced inhibition of degranulation. The data indicate that toxin B inhibits activation of RBL cells by glucosylation of low molecular mass GTP-binding proteins of the Rho subfamily (most likely Cdc42) by a mechanism not involving the actin cytoskeleton.

L6 ANSWER 26 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 7
AN 1996:576231 BIOSIS
DN PREV199799290912
TI Leukocyte-endothelial cell interactions evoked by mast cells.
AU Kubes, Paul [Reprint author]; Granger, D. Neil
CS Immunol. Res. Group, Univ. Calgary Med. Cent., Calgary, Alberta T2N 4N1, Canada
SO Cardiovascular Research, (1996) Vol. 32, No. 4, pp. 699-708.
CODEN: CVREAU. ISSN: 0008-6363.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 23 Dec 1996
Last Updated on STN: 23 Dec 1996

AB In this review we have summarized some of the evidence to support the view that mast cells play a critical role in leukocyte recruitment to sites of inflammation. Initially, data using a pharmacological tool, compound 48/80, which directly activates mast cells, is reviewed, demonstrating that this reagent can induce the multi-step recruitment of leukocytes (rolling, adhesion and emigration) to sites of inflammation. The adhesive mechanisms and pro-inflammatory mediators implicated in mast cell-induced leukocyte recruitment are discussed. Additionally, data are presented to implicate mast cells in delayed-type hypersensitivity reactions as they pertain to leukocyte recruitment. There is a growing body of evidence to suggest that mast cells also recruit leukocytes in IgE -independent leukocyte recruitment. Ischemia/reperfusion- and bacterial toxin- (Helicobacter pylori and Clostridium difficile) induced leukocyte recruitment is at least in part mast cell dependent. Future directions including preliminary work highlighting the role of nitric oxide as a modulator of mast cell function and subsequent leukocyte recruitment is also discussed.

L6 ANSWER 27 OF 31 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN DUPLICATE 8
AN 1996:407385 BIOSIS
DN PREV199699129741
TI Effect of Clostridium difficile toxin B on IgE receptor-mediated signal transduction in rat basophilic leukemia cells: Inhibition of phospholipase D activation.
AU Ojio, Katsuhiro [Reprint author]; Banno, Yoshiko; Nakashima, Shigeru; Kato, Naoki; Watanabe, Kunitomo; Lyerly, David M.; Miyata, Hideo; Nozawa, Yoshinori
CS Dep. Otolaryngology, Gifu Univ. Sch. Med., Tsukasamachi-40, Gifu, Japan
SO Biochemical and Biophysical Research Communications, (1996) Vol. 224, No. 2, pp. 591-596.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 10 Sep 1996
Last Updated on STN: 10 Sep 1996
AB Antigen (Ag)-stimulated phospholipase D (PLD) activation and secretion were almost abolished by pretreatment of rat basophilic leukemia (RBL)-2H3 cells for 4 h with 5 ng/ml Clostridium difficile Toxin B which is known to inhibit Rho family proteins (Rho, Cdc42, Rac). The concentration-dependent inhibition of PLD activation was well correlated with the level of glucosylation of Rho fan-Lily proteins. In streptolysin O-permeabilized RBL cells, Toxin B suppressed (3H) phosphatidylbutanol (PBut) formation in response to guanosine 5'-O-(3-thiotriphosphate) (GTP-gamma-S) and phorbol 12-myristate 13-acetate (PMA) by 67 and 43%, respectively. The synergistic PLD activation by GTP-gamma-S and PMA was also reduced by Toxin B by 67%. These results suggest that the IgE receptor-coupled PLD activation is largely mediated by Rho proteins.

L6 ANSWER 28 OF 31 MEDLINE on STN
AN 96235610 MEDLINE
DN PubMed ID: 8690250
TI Regulation of exocytosis by the small GTP-binding protein Rho in rat basophilic leukemia (RBL-2H3) cells.
AU Yonei S G; Oishi K; Uchida M K
CS Department of Molecular Pharmacology, Meiji College of Pharmacy, Tokyo, Japan.
SO General pharmacology, (1995 Nov) Vol. 26, No. 7, pp. 1583-9.
Journal code: 7602417. ISSN: 0306-3623.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English
FS Priority Journals
EM 199608
ED Entered STN: 11 Sep 1996
Last Updated on STN: 3 Mar 2000
Entered Medline: 27 Aug 1996
AB 1. We investigated the effect of Clostridium botulinum C3 ADP-ribosyltransferase upon beta-hexosaminidase release induced by various stimuli from streptolysin-O (0.5-1 U/ml)-permeabilized rat basophilic leukemia (RBL-2H3) cells. 2. The C3 transferase inhibited beta-hexosaminidase release induced by Ca2+ or by guanosine-5'-(3-thiotriphosphate) (GTP gamma S) plus Ca2+. 3. The C3 transferase also inhibited beta-hexosaminidase release induced by stimulating high affinity IgE and m3 muscarinic acetylcholine receptors. 4. The substrate for the C3 transferase was present in cytosol of RBL-2H3 cells, indicating the presence of rho p21. About 60% of the total cellular substrate protein remained within the cells permeabilized by 1 U/ml of streptolysin-O. 5. The protein rho p21 appears to be regulated by several pathways and it may function as an integration point for exocytosis.

L6 ANSWER 29 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1982:421883 CAPLUS
DN 97:21883
TI Immunoglobulin E-suppressing and immunoglobulin G-enhancing tetanus toxoid prepared by conjugation with pullulan
AU Mitani, Shoko; Yamamoto, Akio; Ikegami, Hakuo; Usui, Mitsuko; Matuhasi, Tyoku
CS Inst. Med. Sci., Univ. Tokyo, Tokyo, 108, Japan
SO Infection and Immunity (1982), 36(3), 971-6
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB IgE antibody response was suppressed selectively and antigen-specifically in mice given an antigen conjugated with pullulan, a linear copolymer of maltotriose, whereas IgM and IgG antibody responses were enhanced. On the basis of this finding, tetanus toxin was conjugated with pullulan by cyanuric chloride in the hope that the toxin would be detoxified by the conjugation procedure and could be used as an IgE-suppressing and IgG-enhancing toxoid without the aid of an Al(OH)3 adjuvant. This procedure of tetanus toxin -pullulan conjugation apparently detoxified the toxin. Administration of the resulting tetanus toxoid (tetanus toxin -pullulan conjugate) to mice induced strong suppression of IgE antibody response with good IgG response, whereas the alum-precipitated toxoid or plain toxoid, customarily used for vaccination, elicited high IgE antibody formation. The IgE antibody response was minimal, but the IgG antibody response was maximal in the conjugate-primed mice even after a booster injection with an IgE antibody-inducing dose of the alum-precipitated toxoid.

L6 ANSWER 30 OF 31 LIFESCI COPYRIGHT 2007 CSA on STN
AN 82:48649 LIFESCI
TI Elevation of levels of IgE antibody to tetanus toxin in individuals vaccinated with diptheria-pertussis-tetanus vaccine.
AU Matuhasi, T.; Ikegami, H.
CS Second Dep. Bacteriol., Natl. Inst. Health, Tokyo, Japan
SO J. INFECT. DIS., (1982) vol. 146, no. 2, pp. 290-291.
DT Journal
FS J; F
LA English
SL English
AB In the present study, levels of IgE antibody to tetanus toxin in the unvaccinated adults were < 100 units/ml (average,

similar to 70 units/ml), levels that were comparable with those of the unvaccinated infants. Levels of IgE antibody were significantly elevated in sera obtained from the infants after vaccination with DPT vaccine. Similar levels were detectable in the sera of the young adults (students from a nursing school). The levels of IgG antibody to tetanus toxin was also elevated in the young adults, although these levels were not elevated to the same extent as the levels of IgE antibody (data not shown). These findings indicate that, after immunization with DPT vaccine, levels of IgE antibody to tetanus toxin are elevated independently of levels of IgG antibody.

L6 ANSWER 31 OF 31 CAPLUS COPYRIGHT 2007 ACS on STN
AN 1981:575785 CAPLUS
DN 95:175785
TI Vaccine based on a biologically active substance joined to a saccharide
IN Matuhashi, Tyoku; Usui, Mitsuko; Yamamoto, Akio; Mitsuhashi, Masakasu; Koyama, Shunsaku
PA Hayashibara Biochemical Laboratories, Inc., Japan
SO Fr. Demande, 18 pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2464074	A1	19810306	FR 1980-19109	19800904
	FR 2464074	B1	19830624		
	JP 56039022	A	19810414	JP 1979-112848	19790905
	JP 57008090	B	19820215		
	US 4372883	A	19830208	US 1980-178904	19800818
	CA 1156553	A1	19831108	CA 1980-358690	19800820
	DE 3032488	A1	19810402	DE 1980-3032488	19800828
	DE 3032488	C2	19880609		
	GB 2061955	A	19810520	GB 1980-28230	19800902
	GB 2061955	B	19830727		
PRAI	JP 1979-112848	A	19790905		

AB Vaccines containing elevated contents of Igs (Ig) G and M and free of IgE and antibodies responsible for allergy and anaphylactic shock were prepared by the inactivation of a biol. toxic substance, e.g. bacterial toxins, by conjugation (covalence) with a saccharide. Thus, an antitetanus vaccine with low toxicity was prepared by conjugating the purified tetanus toxin with BrCN-activated pullulan, mol. weight 140,000. Administration of this vaccine produced .apprx.12 times more IgG and M, compared to the unconjugated toxin with which IgE was detected. Other saccharides used for the conjugation of these toxins were partially-hydrolyzed diazotized pullulan, elsinan, CM-cellulose, gum arabic, and maltotriose.

=> s l4 and ((BoNT)or(CnT)or(TeNT))
L7 0 L4 AND ((BONT) OR(CNT) OR(TENT))

=> s ((BoNT)or(CnT)or(TeNT))
L8 12271 ((BONT) OR(CNT) OR(TENT))

=> d bib ab

L8 ANSWER 1 OF 12271 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2007:450913 BIOSIS
DN PREV200700456296
TI Arsenic contents, and the physical and chemical properties of soil at an arsenic contaminated region in Nepal.
AU Kondo, Fumiyoshi [Reprint Author]; Sugimoto, Yasuhiro; Toyomitsu, Yukio;

Yokota, Hiroshi
CS Saga Univ, Fac Agr, 1 Honjo-machi, Saga 8408502, Japan
SO Transactions of the Japanese Society of Irrigation Drainage and Reclamation Engineering, (APR 2007) Vol. 75, No. 2, pp. 81-87. ISSN: 0387-2335.
DT Article
LA English
ED Entered STN: 22 Aug 2007
Last Updated on STN: 22 Aug 2007
AB The relationships between arsenic content and physical and chemical properties of soil at an arsenic contaminated region in Nepal were investigated. Arsenic content exceeding 150mg/kg was not detected in soils of agricultural land. In this case, it was found out that greater arsenic content occurred concomitantly with soils having higher cation exchange capacity. In boring core samples, accumulated arsenic content exceeding 150mg/kg was detected in the black-colored peat layer found at a depth of 10-11 m. It was assumed that the arsenic which was originally contained in this layer could easily leach into groundwater as a result of activity of microorganisms etc. In addition, it was also found out that greater arsenic con,tent occurred concomitantly with boring core samples having higher clay content, pH,and cation exchange capacity.

=> d kwic

L8 ANSWER 1 OF 12271 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AB. . . into groundwater as a result of activity of microorganisms etc. In addition, it was also found out that greater arsenic con,tent occurred concomitantly with boring core samples having higher clay content, pH,and cation exchange capacity.

=> s ((BoNT)or(CnT))
L9 7792 ((BONT) OR(CNT))

=> d kwic

L9 ANSWER 1 OF 7792 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AB. . . unique strain of Clostridium botulinum serotype D 4947 produces toxin complexes that are composed of un-nicked components, including a neurotoxin (BoNT) and auxiliary proteins. This BoNT showed aberrant elution upon Superdex gel filtration, indicating a much lower molecular weight, due to hydrophobic interaction with the column. Limited trypsin proteolysis of BoNT produces two nicks; first nick yielded a BoNT 50 kDa light chain disulfide linked to a 100 kDa heavy chain (Hc), and a second nick arose in Hc C-terminal 10 kDa. The second nick occurred in the putative binding domain of the BoNT molecule and induced alterations in its secondary structure, leading to a significant reduction of mouse toxicity in comparison with that of the fully-activated singly nicked BoNT. These results help to clarify the role of the C-terminal half of the Hc in the oral toxicity of single-chain and more complex forms of BoNT.

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L9 ANSWER 1 OF 7792 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN
AN 2007:440396 BIOSIS
DN PREV200700440956
TI Effect of nicking the c-terminal region of the Clostridium botulinum serotype d neurotoxin heavy chain on its toxicity and molecular

properties.
AU Suzuki, Tomonori; Kouguchi, Hirokazu; Watanabe, Toshihiro; Hasegawa, Kimiko; Yoneyama, Tohru; Niwa, Koichi; Nishikawa, Atsushi; Lee, Jae-Chul; Oguma, Keiji; Ohyama, Tohru [Reprint Author]
CS Tokyo Univ Agr, Fac Bioind, Dept Food Sci and Technol, 196 Yasaka, Abashiri, Hokkaido 0992493, Japan
t-oyama@bioindustry.nodai.ac.jp
SO Protein Journal, (APR 2007) Vol. 26, No. 3, pp. 173-181.
ISSN: 1572-3887.
DT Article
LA English
ED Entered STN: 15 Aug 2007
Last Updated on STN: 15 Aug 2007

=> s l9 and IgE
L10 3 L9 AND IGE

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L10 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2006:1291807 CAPLUS
DN 146:201074
TI Label-Free Protein Biosensor Based on Aptamer-Modified Carbon Nanotube Field-Effect Transistors
AU Maehashi, Kenzo; Katsura, Taiji; Kerman, Kagan; Takamura, Yuzuru; Matsumoto, Kazuhiko; Tamiya, Eiichi
CS Institute of Scientific and Industrial Research, Osaka University, Osaka, 567-0047, Japan
SO Analytical Chemistry (2007), 79(2), 782-787
CODEN: ANCHAM; ISSN: 0003-2700
PB American Chemical Society
DT Journal
LA English
AB The authors have fabricated label-free protein biosensors based on aptamer-modified carbon nanotube field-effect transistors (CNT -FETs) for the detection of IgE. After the covalent immobilization of 5'-amino-modified 45-mer aptamers on the CNT channels, the elec. properties of the CNT-FETs were monitored in real time. The introduction of target IgE at various concns. caused a sharp decrease in the source-drain current, and a gradual saturation was observed at lower concns. The amount of the net source-drain current before and after IgE introduction on the aptamer-modified CNT-FETs increased as a function of IgE concentration The detection limit for IgE was determined as 250 pM. The authors have also prepared CNT-FET biosensors using a monoclonal antibody against IgE (IgE-mAb). The elec. properties of the aptamer- and antibody-modified CNT-FETs were compared. The performance of aptamer-modified CNT-FETs provided better results than the ones obtained using IgE-mAb-modified CNT-FETs under similar conditions. Thus, the authors suggest that the aptamer-modified CNT-FETs are promising candidates for the development of label-free protein biosensors.
RE.CNT 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB The authors have fabricated label-free protein biosensors based on aptamer-modified carbon nanotube field-effect transistors (CNT -FETs) for the detection of IgE. After the covalent immobilization of 5'-amino-modified 45-mer aptamers on the CNT channels, the elec. properties of the CNT-FETs were monitored in real time. The introduction of target IgE at various concns. caused a sharp decrease in the source-drain current, and a gradual saturation was observed at lower concns. The amount of the net source-drain current

before and after IgE introduction on the aptamer-modified CNT-FETs increased as a function of IgE concentration The detection limit for IgE was determined as 250 pM. The authors have also prepared CNT-FET biosensors using a monoclonal antibody against IgE (IgE-mAb). The elec. properties of the aptamer- and antibody-modified CNT-FETs were compared. The performance of aptamer-modified CNT-FETs provided better results than the ones obtained using IgE-mAb-modified CNT-FETs under similar conditions. Thus, the authors suggest that the aptamer-modified CNT-FETs are promising candidates for the development of label-free protein biosensors.

IT Antibodies and Immunoglobulins
RL: ANT (Analyte); ANST (Analytical study)
(IgE; label-free protein biosensor based on aptamer-modified carbon nanotube field-effect transistors)

L10 ANSWER 2 OF 3 MEDLINE on STN
AN 2007064093 MEDLINE
DN PubMed ID: 17222052
TI Label-free protein biosensor based on aptamer-modified carbon nanotube field-effect transistors.
AU Maehashi Kenzo; Katsura Taiji; Kerman Kagan; Takamura Yuzuru; Matsumoto Kazuhiko; Tamiya Eiichi
SO Analytical chemistry, (2007 Jan 15) Vol. 79, No. 2, pp. 782-7.
Journal code: 0370536. ISSN: 0003-2700.
CY United States
DT Letter
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200703
ED Entered STN: 3 Feb 2007
Last Updated on STN: 24 Mar 2007
Entered Medline: 22 Mar 2007
AB We have fabricated label-free protein biosensors based on aptamer-modified carbon nanotube field-effect transistors (CNT-FETs) for the detection of immunoglobulin E (IgE). After the covalent immobilization of 5'-amino-modified 45-mer aptamers on the CNT channels, the electrical properties of the CNT-FETs were monitored in real time. The introduction of target IgE at various concentrations caused a sharp decrease in the source-drain current, and a gradual saturation was observed at lower concentrations. The amount of the net source-drain current before and after IgE introduction on the aptamer-modified CNT-FETs increased as a function of IgE concentration. The detection limit for IgE was determined as 250 pM. We have also prepared CNT -FET biosensors using a monoclonal antibody against IgE (IgE-mAb). The electrical properties of the aptamer- and antibody-modified CNT-FETs were compared. The performance of aptamer-modified CNT-FETs provided better results than the ones obtained using IgE-mAb-modified CNT-FETs under similar conditions. Thus, we suggest that the aptamer-modified CNT-FETs are promising candidates for the development of label-free protein biosensors.
AB We have fabricated label-free protein biosensors based on aptamer-modified carbon nanotube field-effect transistors (CNT-FETs) for the detection of immunoglobulin E (IgE). After the covalent immobilization of 5'-amino-modified 45-mer aptamers on the CNT channels, the electrical properties of the CNT-FETs were monitored in real time. The introduction of target IgE at various concentrations caused a sharp decrease in the source-drain current, and a gradual saturation was observed at lower concentrations. The amount of the net source-drain current before and after IgE introduction on the aptamer-modified CNT-FETs increased as a

function of IgE concentration. The detection limit for IgE was determined as 250 pM. We have also prepared CNT -FET biosensors using a monoclonal antibody against IgE (IgE-mAb). The electrical properties of the aptamer- and antibody-modified CNT-FETs were compared. The performance of aptamer-modified CNT-FETs provided better results than the ones obtained using IgE-mAb-modified CNT-FETs under similar conditions. Thus, we suggest that the aptamer-modified CNT-FETs are promising candidates for the development of label-free protein biosensors.

L10 ANSWER 3 OF 3 MEDLINE on STN
AN 2006463358 MEDLINE
DN PubMed ID: 16886472
TI [Concentration of allergic fungi spores in the air of flats in Lodz]. Stezenie zarodnikow grzybow alergogennych w powietrzu mieszkan w Lodzi.
AU Krawczyk P; Kowalski M L; Ochecka-Szymanska A
CS Studenckie Kolo Naukowe przy Katedrze Biologii i Parazytologii Lekarskiej AM 90-436 Lodz.
SO Wiadomosci parazytologiczne, (1999) Vol. 45, No. 2, pp. 255-62. Journal code: 0420554. ISSN: 0043-5163.
CY Poland
DT (ENGLISH ABSTRACT) Journal; Article; (JOURNAL ARTICLE)
LA Polish
FS Priority Journals
EM 200611
ED Entered STN: 5 Aug 2006
Last Updated on STN: 19 Dec 2006
Entered Medline: 30 Nov 2006
AB The real contribution of moulds to the pathogenesis of allergic diseases remains unknown, although positive skin prick tests and/or specific serum IgE to moki allergens can be detected in 1-5% of atopic patients. A significant problem in assesment of exposure to mould allergens, resulting with difficulty in standarization of methods. The aim of this work was to assess the concentration of spores of 8 mould species in flats inhabited by peoples who Bont show any symptoms of allergy. The Open Petri Dish (OPD) method involving sedimentation of participles contained in the column of air over the dish was used to assess the number of spores in 1 m3 of indoor atmospheres. All colonies were counted, but only 8 mould species implicated in inhaled allergy were identified, ie.: Alternaria tenuis, Cladosporium herbarum, Helminthosporum halodes, Pullularia pullulans, Penicillium notatam, Rhizopus nigricans, Mucor mucedo, Aspergillus fumigatus. The tests were carried out in 10 flats located in various quarters of the cify of Lodz during three consecutive days of September 1995 between 5:00 pm and 6:04 pm. In analyzing the percentage of spores of each of the eight mould species tested we determined that, independent of fiat and test day, C. herbarum predominated. It is good agreement with the observations of other authors who report that among large quantities of fungi that are detected in late summer, usually C. herbarum spores dominate. This is the season when the incidence of the Cladosporium spores in the atmospheric air increases. Spores of H. halodes were detected least frequently. Our study demonstrated the presence of substantial amounts of mould spores in indoor air of houses in Lodz. The spores belong to species with documented allergenicity, suggesting that they may play a role in development of allergic sensitization in susceptible subjects.
AB . . . real contribution of moulds to the pathogenesis of allergic diseases remains unknown, although positive skin prick tests and/or specific serum IgE to moki allergens can be detected in 1-5% of atopic patients. A significant problem in assesment of exposure to mould. . . of this work was to assess the concentration of spores of 8 mould species in flats inhabited by peoples who Bont show any symptoms of allergy. The Open Petri Dish (OPD) method involving sedimentation of

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